CHAPTER 38

Shortness of Breath

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The patient with acute shortness of breath (dyspnea) presents an immediate challenge to the family physician. Quick assessment, including a rapid airway, breathing, and circulation (ABC) check must first be done, followed by consideration of potentially life-threatening causes. Patients with severe dyspnea may present with chest pain, difficulty speaking, tachypnea, wheezing, rales, accessory muscle use, and hypoxemia. Once the patient is stabilized (i.e., airway secured, oxygen placed, IV established, and cardiac monitor applied), a more thorough history, physical, and laboratory evaluation can be performed to verify the diagnosis.

This chapter focuses on the management of six common diseases that can cause acute dyspnea, including several diagnoses often seen in children (croup and bronchiolitis). Note that there are many causes of chronic dyspnea (duration greater than 3 weeks). These include conditions such as interstitial lung disease and neuromuscular and chest wall disorders. These problems are not discussed in this chapter.

PNEUMOTHORAX

Pneumothorax is the accumulation of air between the two parietal surfaces (visceral and parietal) of the lung. If a check valve mechanism occurs in which the air cannot escape, a tension pneumothorax will develop and cause hemodynamic compromise and acute respiratory failure unless promptly treated with needle aspiration or chest tube placement. Spontaneous pneumothorax (SP) may be primary (PSP) or secondary (SSP). Primary pneumothorax occurs most commonly in tall, thin men (ages 20 to 40). SSP is a complication of diseases such as COPD, cystic fibrosis, and *Pneumocystis jiroveci* (previously *P. carinii*) pneumonia in immunocompromised patients. Iatrogenic (i.e., after central line placement) and trauma are other causes of pneumothorax.

Symptoms

- Shortness of breath +++
- Pleuritic chest pain ++++
- Cough ++
- Anxiety

Signs

- Patients with minimal pneumothoraces may have no detectable signs.
- Tachycardia (most common)
- Tachypnea
- Diminished breath sounds on affected side
- Hyperresonance to percussion
- Subcutaneous emphysema
- Neck vein distention, tracheal deviation with tension pneumothorax

Workup

- Patients with large or tension pneumothorax should be treated with needle or tube thoracostomy prior to any investigations.
- Pulse oximetry, consider ABG
- ECG monitoring, IV placement
- Chest radiograph: look for displacement of visceral line from chest wall
- CT scan may be needed to distinguish bullae from pneumothorax in COPD patients

Comments and Treatment Considerations

There is little evidence from randomized trials to support management decisions. Consensus statements (expert opinion) from American College of Chest Physicians and the British Thoracic Society are the best available evidence. Goals of therapy are to eliminate air in the pleural space and prevent recurrence.

Most patients will require subspecialty assistance from a pulmonologist or surgeon. High-flow oxygen therapy will increase resorption of pleural space air (exercise caution with COPD patients who retain CO_2). Management decisions depend on the size of the pneumothorax and clinical stability of the patient. If the patient is clinically stable and lung collapse is less than 2 to 3 cm, observation may be sufficient. Larger pneumothoraces and unstable patients often require aspiration or chest tube placement.

Patients with SSP tend to be more symptomatic, and frequently require hospitalization. Observation and oxygen therapy alone are usually not sufficient. Furthermore, aspiration is less likely to succeed in these patients—most will need chest tubes. Complicated patients with air leaks, bronchopleural fistulas, and recurrent pneumothoraces need subspecialty attention.

Most patients with PSP who have a second occurrence will require thoracoscopy or thoracotomy for prevention. Chemical pleurodesis with talc or doxycycline is considered second-line therapy. Patients with SSP may need surgical intervention after the first pneumothorax. For tension pneumothorax insert a 19-gauge needle into the second intercostal space at the midclavicular line over the superior aspect of the rib. Attach a three-way stopcock, and withdraw air with a large syringe. Then insert a chest tube after the patient is stabilized.

All patients should be encouraged to avoid or reduce risk factors, with particular emphasis on smoking cessation.

CONGESTIVE HEART FAILURE

Heart failure is classified as systolic or diastolic dysfunction. Systolic dysfunction can occur whenever changes affect heart rate, contractility, preload or afterload. The most common cause of acute systolic dysfunction is loss of contractility due to MI. Valvular disease, dysrhythmias, and severe hypertension can contribute to pump failure. Heart failure can present in the face of normal left ventricular systolic function. Diastolic dysfunction is a result of a stiff or noncompliant ventricle resulting in abnormal filling of the ventricle. This can lead to elevated diastolic pressures and a diminished cardiac output. Chronic long-standing hypertension is a common cause of diastolic dysfunction. A variety of neurohormonal and hemodynamic changes occur in response to a decrease in cardiac output that results in vasoconstriction and salt and water retention, which worsens both preload and afterload. This cycle leads to further decreases in cardiac output.

Symptoms

- Dyspnea with exertion ++++
- Fatigue
- Cough
- Angina if coexisting coronary artery disease
- Wheezing
- Orthopnea ++
- Paroxysmal nocturnal dyspnea +++

Signs

- Tachycardia
- Tachypnea
- Peripheral edema ++ (85% specific)
- Anxiety
- Frothy, blood-tinged sputum if acute pulmonary edema
- Moist rales on auscultation
- Wheezes with bronchospasm
- Jugular venous distention ++
- Murmur(s)
- S_3 gallop ++ (90% specific)
- S_4 gallop (especially in diastolic dysfunction)
- Hypoxia

Workup

- Chest radiograph
- ECG
- Pulse oximetry, consider ABG
- Chemistry (assess renal function, potassium, and liver enzymes)
- CBC
- BNP is helpful for distinguishing between pulmonary and cardiac causes in patients with coexisting CHF and lung disease.
- TSH
- Cardiac enzymes
- Echocardiogram (after stabilization)

Comments and Treatment Considerations

Preload Reduction

Diuretics have been shown to reduce both mortality and readmission for worsening heart failure symptoms (level 1A). Nitroglycerin is thought to be the fastest-acting, most effective, and predictable agent for preload reduction. Be cautions not to overdiurese patients with diastolic dysfunction or mainly right-side failure. This can cause hypotension and electrolyte derangements. Aldosterone antagonists such as spironolactone have been shown to reduce mortality, death and hospitalization from cardiac causes and death from CHF, and improved New York Heart Association (NYHA) functional class in patients with severe CHF (level 1A). Morphine sulfate's main effects are likely secondary to reduced anxiety and air hunger, which can decrease catecholamine production and thereby help reduce systemic vascular resistance.

Afterload Reduction

ACE inhibitors appear to decrease mortality in heart failure patients. They should be used with caution in patients with borderline hemodynamic status. Evidence shows that these ARBs are a good choice for patients who are ACE intolerant, and may be used in combination with ACE inhibitors. Hydralazine used either alone or with nitrates is an option in patients with renal insufficiency or ACE intolerance. One trial showed improved survival and quality of life with isosorbide dinitrate and hydralazine in African-American patients.

Beta-Blockers

Once heart failure is stabilized, beta-blockers such as carvedilol and extended-release metoprolol have been shown to decrease mortality.

Inotropic Agents

Inotropic agents may be necessary if a patient remains symptomatic. There is no survival benefit with digoxin. Digoxin can be helpful for rate control in patients with tachyarrhythmias as well. Dobutamine and dopamine are other choices for inotropic support.

Nesiritide

Human B-type natriuretic peptide, after review of evidence, needs more study. One review showed a trend toward increased mortality in acute decompensated heart failure.

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

COPD is a chronic disorder associated with airflow obstruction usually caused by chronic bronchitis or emphysema. The definition of chronic bronchitis requires the presence of a chronic cough and/or production of sputum for 3 months each year over 2 consecutive years. Emphysema is characterized by abnormal, permanent enlargement of the air spaces distal to the terminal bronchioles, with destruction of the walls, and without fibrosis. Although emphysema is

usually described anatomically, and chronic bronchitis is described clinically, both are often seen together. There can be airway hyperreactivity and small airway inflammation that may be partially reversible. Acute exacerbation of symptoms occurs more frequently as the disease progresses and is a common cause of acute dyspnea.

Symptoms

- Shortness of breath
- Productive cough with increase in sputum production during acute exacerbation
- Wheezing

Signs

- Tachypnea
- Tachycardia
- Accessory muscle use
- Coarse rhonchi
- Hyperresonance to percussion
- Bilateral decreased breath sounds
- Hyperinflation (barrel chest)
- Prolonged expiratory phase
- Pursed-lip breathing
- Cyanosis

Workup

- Chest radiograph
- Pulse oximetry, consider ABG
- BNP is helpful for distinguishing between pulmonary and cardiac causes in patients with coexisting CHF (level 1B).
- CBC with differential
- Serum electrolytes
- Spirometry if not done previously (FEV_1/FVC <0.70 indicates airflow obstruction)
- α_1-antitrypsin if history suggestive

Comments and Treatment Considerations

Oxygen should be used to correct hypoxia. Most cases of CO_2 retention are due to ventilation-perfusion mismatch and not depression of the respiratory center. Therefore the correction of hypoxemia should be the therapeutic preference. Oral or parenteral corticosteroids can reduce treatment failures and the need for further medical treatment.

Antibiotics reduce risk of short-term mortality by 77%, treatment failure by 53%, and sputum purulence by 44%. Inhaled bronchodilators are used routinely. The co- administration of β2 agonists and ipratropium bromide seem to be more effective than either one alone.

In patients with acute respiratory failure, noninvasive positive pressure ventilation (NPPV) can reduce likelihood of endotracheal intubation, treatment failures, and mortality. It has also been shown to reduce length of hospital stay and complications associated with treatment.

ASTHMA

Asthma is an obstructive lung disease characterized by acute, intermittent periods of tracheobronchial hyper-responsiveness to a variety of stimuli. The reversibility of the acute, obstructive attack and recurrence is diagnostic of this chronic pulmonary disease. In an acute setting, management of the acute exacerbation requires timely clinical assessment and treatment to improve airflow and decrease inflammation. Respiratory distress that is unsuccessfully managed (status asthmaticus) may lead to death. Goals of chronic asthma therapy include patient education, periodic assessment of pulmonary function, identification and avoidance of patient-specific triggers, and the use of pharmacologic agents that decrease airway inflammation and provide airway bronchodilation.

Symptoms

- Wheezing ++++
- Daytime and/or nighttime cough +++
- Shortness of breath +++
- Chest tightness
- Symptoms exacerbated by illness, exercise, or environmental exposure

Signs

- Wheezing
- Intercostal retractions and use of accessory muscles
- Prolonged expiratory phase
- Tachypnea
- Inability to speak in complete sentences
- Nasal obstruction, including nasal polyps
- Atopic dermatitis or eczema

Workup

- Peak flow meter
- Spirometry (FEV_1/FVC <0.70 indicates airflow obstruction)
- Arterial blood gas
- Chest radiograph
- Exercise spirometry

Comments and Treatment Considerations

Severity of disease varies with frequency of symptoms and FEV_1. For intermittent asthma symptoms will last 2 or fewer days per week, or 2 or fewer night awakenings per month and FEV_1 80% or more predicted value. Treat with short-acting bronchodilators as needed for acute exacerbation. Daily therapy is not indicated.

Mild persistent asthma is defined as symptoms occurring more than 2 days per week—but not daily—(or more than 2 night awakenings per month) and FEV_1 80% or more predicted value. Treatment includes short-acting bronchodilators as needed for acute exacerbation and low-dose inhaled corticosteroids for daily antiinflammatory management.

Moderate persistent asthma has daily symptoms, or more than 1 night awakening per week and FEV_1 60% or more, but less than 80% predicted value. Treat with short-acting bronchodilators as needed for acute exacerbation and daily medium-dose inhaled corticosteroids and (if required) long-acting bronchodilator.

Severe persistent asthma will have continual daytime symptoms, or nightly symptoms and FEV_1 60% or less predicted value. Treat with short-acting bronchodilators as needed for acute exacerbation. Long-term therapy should include a medium- or high-dose inhaled corticosteroid, in addition to a long-acting bronchodilator and oral corticosteroids.

Status asthmaticus has prolonged exacerbations of symptoms and fails to respond to usual treatment. Peak expiratory flow is less than 50% of predicted or best effort, oxygen saturation is less than 91% and Pao_2 less than 60 mm Hg on room air or Pco_2 greater than 42 mm Hg. Short-acting bronchodilators, inhaled ipratropium bromide, supplemental oxygen and corticosteroids are the mainstays of medical management. Hospitalize patients that do not demonstrate immediate improvement of the acute exacerbation. Consider subcutaneous and IV epinephrine for severely ill patients. A one-time bolus of intravenous magnesium sulfate may be considered for those with impending respiratory failure. Prepare to intubate any patient with progressively worsening respiratory distress, especially if patient appears to be fatiguing. However, intubation should be accomplished as a last resort because air trapping makes ventilation extremely difficult after intubation.

Other Considerations

Use of inhaled corticosteroids at the lowest effective doses offers the maximum benefit and fewest adverse effects.

Children with mild to moderate asthma demonstrate better control of their disease when using inhaled corticosteroids than with use of β-agonists as a single agent.

Although a short-term difference in growth velocity (1 cm/yr) has been observed in children with chronic use of inhaled corticosteroids, the difference does not affect final adult height.

Although written action plans for asthma management in children are recommended, there is limited evidence that symptom-based plans offer significant advantage over peak flow–based plans.

Antibiotics do not offer any benefit in the management of acute asthma exacerbation in the absence of a bacterial infection of the respiratory system.

The role of leukotriene modifiers in the chronic management of asthma is unclear. Patients with aspirin-sensitive asthma seem to derive the most benefit from this medication class.

BRONCHIOLITIS

Bronchiolitis is the most common lower respiratory tract illness of infants and very young children. It is a clinical syndrome characterized by wheezing and tachypnea. RSV causes the majority

of cases; others are caused by other respiratory viruses. Peak incidence in the United States occurs in the winter and early spring. Patients at highest risk for severe disease include premature infants; infants less than 6 weeks old; those with chronic lung, heart, or neurologic disease; and those suffering from immunodeficiency.

Symptoms
- Cough
- Dyspnea
- Poor feeding

Signs
- Wheezing with or without rales
- Rhinorrhea
- Tachypnea
- Fever
- Retractions
- Nasal flaring
- Decreased oxygen saturation
- Cyanosis
- Apnea

Workup
- History and physical examination provide basis for diagnosis. Accurate assessment of severity is critical. Patients at highest risk for adverse outcomes include those with chronic lung disease, prematurity, congenital heart disease, and immunocompromise.
- Routine diagnostic studies (RSV swab, chest x-ray, cultures, or ABGs) not indicated in low-risk cases of mild severity
- Chest x-rays may be useful when diagnosis is unclear or the patient is not improving as expected.
- Rapid viral testing may be beneficial in avoidance of unnecessary testing in very young (<3 months) infants.

Comments and Treatment Considerations
Primary treatment is supportive. Consider hospitalization for infants with respiratory rates of 50 or more and/or saturations less than 95% on room air. Assess for and treat dehydration. Suctioning should be done when needed, before feeding, and prior to inhalation therapy.

Results of trials of nebulized β-agonists or racemic epinephrine have been equivocal. Many clinicians will attempt a trial of these agents early in the course of the illness. Repeated clinical assessment should be done for signs of deteriorating respiratory status. Mechanical ventilation is indicated for respiratory failure.

The use of ribavirin is controversial, its use is generally reserved for selected immunocompromised patients with severe disease. Prophylaxis with palivizumab is recommended in certain target groups, including infants with chronic lung disease, prematurity, or congenital heart disease. Antibiotics should be used only if there is clear evidence of a coexisting bacterial infection.

CROUP

Croup is a clinical syndrome seen most commonly in children between the ages of 3 months and 3 years. Croup is characterized by inspiratory stridor, a brassy or barklike cough, hoarseness, and varying degrees of respiratory distress. These symptoms are secondary to inflammation and edema of the laryngeal mucosa leading to subglottal narrowing. Bacterial tracheitis may be difficult to distinguish from croup, but typically the child with tracheitis appears more toxic. Foreign body aspiration may also present with croup-like symptoms. Acute epiglottitis and retropharyngeal abscess classically lack the barking cough of croup but may present with stridor, fever, and labored breathing.

Symptoms

- A barklike or brassy cough is a classic symptom. ++++
- Hoarseness
- Inspiratory stridor
- Symptoms worse at night
- Rhinorrhea, nasal congestion
- Irritability, poor feeding

Signs

- Inspiratory stridor
- Barklike cough ++++
- Mild to moderately inflamed pharynx
- In-drawing of chest wall with inspiration
- Respiratory rate may be increased
- Fever may be present; less likely with spasmodic croup
- Hypoxia in severe cases

Workup

- Diagnosis is clinical.
- Lateral neck x-ray may show subglottic narrowing (steeple sign).
- Pulse oximetry is normal in most, but should be used in monitoring those with more severe croup.
- Use caution in getting lab work or x-rays (if indicated) because agitation of the patient may increase respiratory distress.

Comments and Treatment Considerations

- Avoid agitation to minimize respiratory distress.
- A single dose of corticosteroids is beneficial regardless of clinical severity.
- Standard steroid treatment for croup is a single dose of dexamethasone 0.6 mg/kg PO to a maximum of 10 mg (or IM if unable to take PO).
- Inhaled budesonide is an alternative to IM dexamethasone in a vomiting child.
- Nebulized racemic epinephrine improves croup symptoms within 10 to 30 minutes, and can be repeated. Duration of improvement is about 2 hours.

- Children given one dose of steroid and a treatment of nebulized racemic epinephrine in the ED can be considered for discharge if symptoms have not recurred by 4 hours post treatment.
- Indications for hospitalization include persistent stridor at rest, hypoxemia, pallor or cyanosis, respiratory distress, depressed level of consciousness, or suspected epiglottitis.

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